AMENDMENTS TO THE SPECIFICATION

Please amend the specification as follows:

Page 3, paragraph [0019]:

The patent or application file contains at least one drawing executed in color. Copies of this patent or patent application publication with color drawing(s) will be provided by the Office upon request and payment of the necessary fee. **FIG.1** is a schematic of an exemplary IKK2 pathway specific reporter assay.

Page 5, paragraph [0048]:

However, in especially preferred aspects of the inventive subject matter, the expression cassette is stably transfected into the assay cell genome and expression of the kinase of interest is under the control of an inducible promoter, wherein the promoter does not naturally occur in the assay cell. For example, expression may be driven from the commercially available ~~Tet-On/Off~~ **TET-ON® / TET-OFF®**. expression system (e.g., by Invitrogen), which employs a chimeric transactivator to regulate transcription of the gene of interest (here: the kinase of interest) from a silent promoter. The transactivator, either tTA or rtTA, is expressed from a constitutive CMV promoter present in the expression cassette (alternatively, the transactivator can also be expressed from a tissue-specific promoter). In the BD ~~Tet-On™~~ **TET-ON®** System, rtTA binds to the TRE (Tetracycline Response Element) and activates transcription in the presence of doxycycline, which is a synthetic analog of Tetracycline. Without doxycycline, substantially no recombinant protein is expressed.

Page 5, paragraph [0055]:

A first portion of the so transformed founder cells is then transformed with a second expression cassette to provide inducible expression of MEK* (constitutively active form of MEK) as the first kinase of the MEK-ERK kinase signal transduction pathway. Similarly, a second portion of the transformed founder cells is transformed with another second expression cassette to provide inducible expression of ERK* (constitutively active form of ERK) as the second kinase of the MEK-ERK kinase signal transduction pathway. Preferred expression cassettes for the kinases are ~~Tet-On™~~ **TET-ON®** expression cassettes as the expression of the kinases can be induced via doxycycline.

Page 8, paragraph [0077]:

Cells that contain inducible wild type or mutant MEK (referred to as MEK*) were generated by transfecting 293T via lipid-mediated gene transfer of the following plasmids. Plasmids for the Tet repressor (Invitrogen pCDNA6-TR), Gal4-Elk1 (Stratagene), Gal4 UAS dependent luciferase (Stratagene), and Tet-On MEK or Tet-On MEK* (Invitrogen) were transfected into cells and the cells placed under selection in 500 ug/ml geneticin, 100 ug/ml zeocin, and 2.5 ug/ml blasticidin. Tet-On MEK was created by placing the cDNA sequence for human MEK under the control of the ~~Tet-On~~ TET-ON® promoter in pCDNA4TO (Invitrogen). ~~Tet-On~~ TET-ON® MEK* was created by site-directed mutagenesis of S218 and S222 to aspartic acids in the human MEK cDNA sequence. Figure 5 depicts an autoradiograph (ECL) showing expression of MEK/MEK* in 293T cells.

Page 8, paragraph [0078]:

Upon establishing ~~Tet-On~~ TET-ON® MEK and ~~Tet-On~~ TET-ON® MEK* clones, we tested the ability of doxycycline to induce gene expression that resulted in ERK phosphorylation and activation was tested. These clones were tested by plating cells from the respective clones and adding lug/ml doxycycline for the times indicated. Cells were lysed and the proteins run on an SDS-PAGE gel. Proteins were transferred to a membrane and the levels of ERK phosphorylation were detected by Western blotting with anti-phospho-ERK antibodies (Cell Signaling Technologies). The presence of phospho-ERK was visualized through attachment of a secondary antibody (goat-anti-rabbit) conjugated to alkaline phosphatase (AP). AP can cleave substrates thus releasing light to be captured on film or by a scanner as depicted in Figure 6.